

Case Report

Radiofrequency Catheter Ablation with the Use of a Noncontact Mapping System for Ventricular Tachycardia Originating from the Aortic Sinus Cusp —A Case Report—

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Here we present a 15-year old female in whom an idiopathic ventricular tachycardia (VT) originating from the left aortic sinus cusp was eliminated by radiofrequency catheter ablation (RFCA) under navigation using a noncontact mapping system (NCM). The dynamic activation map constructed with the NCM clearly identified a VT focus in the left aortic sinus cusp, from which the activation spread out to the entire left ventricle. At that site, the virtual unipolar electrogram recorded with the NCM was the same as the contact unipolar electrogram in terms of morphology and timing, a pre-systolic potential preceding the QRS complex by 40 msec was recorded by contact bipolar electrogram and rapid pacing during sinus rhythm resulted in a perfect pace match. RFCA at that site eliminated the VT and the patient has had no recurrence during 10 months of follow-up.
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Key words: Aortic sinus cusp, Noncontact mapping, Ventricular tachycardia

Introduction

The noncontact mapping system (NCM) has provided us with a deep insight into mechanisms of tachycardia irrespective of the focal or reentrant mechanism.^{1–8)} However, the efficacy of the non-

contact mapping system in radiofrequency catheter ablation (RFCA) of idiopathic ventricular tachycardia (VT) is still unclear. Here we describe a case in which an idiopathic VT originating from the left aortic sinus cusp (ASC) was eliminated by RFCA under navigation using the NCM.

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Case Report

A 15-year old female was referred for RFCA of a sustained VT, which was documented at medical check up. The electrocardiogram exhibited a left bundle branch block and inferior axis pattern, suggesting that the VT originated from the left coronary cusp or superior portion of the mitral annulus (**Figure 1**). The patient complained of slight palpitations, and exercise limitations. No organic heart disease was documented by a 12-lead electrocardiography recorded during sinus rhythm, echocardiography, coronary angiography, or left ventriculography. In the Holter electrocardiographic recordings, repetitive VT with a maximum of 813 successive beats were documented and accounted for >60% of the total heart beats in a single day. In this case, medical treatment could be considered as an alternative, but both the patient and her parents expressed a preference for RFCA.

Written informed consent was obtained and the session was performed under conscious sedation with a small amount of fentanyl and midazolam. The basic rhythm was nonsustained VT, the same QRS morphology which was documented during the palpitation attacks.

A multielectrode array (MEA) used with the NCM (St. Jude Medical, Minnetonka, MN, USA) was introduced into the left atrium (LA) through a long transseptal sheath which was advanced into the LA by the standard Brockenbrough technique. Then the MEA was slightly advanced into the left ventricle (LV) so as to locate one fourth of the MEA at the junction between the LA and LV. Throughout the study, ACT was monitored every 30 minutes and, if it was below 300 seconds, an adequate amount of heparin was injected to maintain ACT between 300 to 400 seconds. The geometry of the LV and ASC was depicted with the ablation catheter advanced via a trans-aortic approach. The recording and analysis

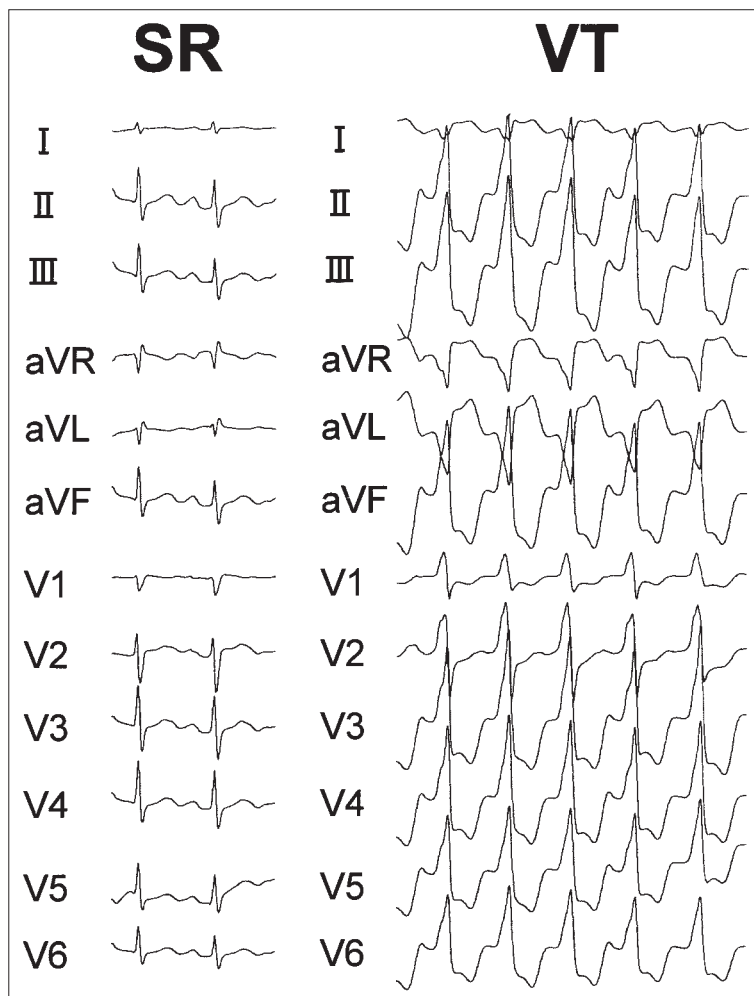


Figure 1 Twelve-lead EKG during sinus rhythm (SR) and the ventricular tachycardia (VT).

No significant abnormalities were detected during SR. The VT cycle length was 464 msec. The axis of the VT was inferior and had a rightward deviation. Note that R is >S in lead V1 and that the S wave cannot be seen in leads V5-6. These findings imply that the VT focus was located at the left coronary cusp or superior portion of the mitral annulus.

by the NCM was performed according to an established method reported by other researchers; the details of NCM have been described elsewhere.¹⁻⁸⁾ During the review of the recorded data, a high-pass filter setting of 8.0 Hz was used for the isopotential map created by the NCM.⁸⁾

During the dynamic wavefront map of the LV and ASC, the earliest activation was identified at the left coronary cusp (LCC), from which the activation proceeded slowly toward the LV septum, and then rapidly to the entire LV. The virtual unipolar electrogram (VUE) at the site exhibiting a QS pattern (Figure 2), the onset of which corresponded to that of the pre-systolic potential recorded by a contact bipolar electrogram, preceded the QRS complex of the VT by 40 msec. It was noted that the morphology and timing of the contact unipolar electrogram were the same as the VUE obtained by the NCM, which validated the VUE. Ventricular pacing which was performed at a stimulus strength of twice the diastolic threshold and with a pulse width of 2 msec using a programmable stimulator (SEC-3102, Nihon Kohden, Japan) did not initiate and terminate the VT, and ventricular pacing during VT did not exhibit an entrainment phenomenon.

Pacing during sinus rhythm at that site (9.9 V) exhibited a perfect pace match as shown in Figure 3. The time interval from the stimulus artifact to the QRS complex during the pacing was 0 msec. Before the RFCA, left coronary angiography was performed in order to confirm that the site was at least more than 1 cm from the left coronary artery orifice (Figure 4b-1, 2). A radiofrequency energy application with an energy output of 20 W (the upper temperature limit was 50 °C) resulted in the immediate termination of the VT, while a non-clinical VT and/or frequent premature ventricular contractions were induced by the accidental stimulation with a guidewire introduced through the MEA into the LV (Figure 4c). After the ablation, the VT could no longer be induced by ventricular pacing and/or an isoproterenol injection. There were no procedural complications such as thromboembolism, or mitral/aortic insufficiency. During a follow-up period of 10 months, the patient has been free from any VT recurrence.

Discussion

VT originating from the ASC has been reported to

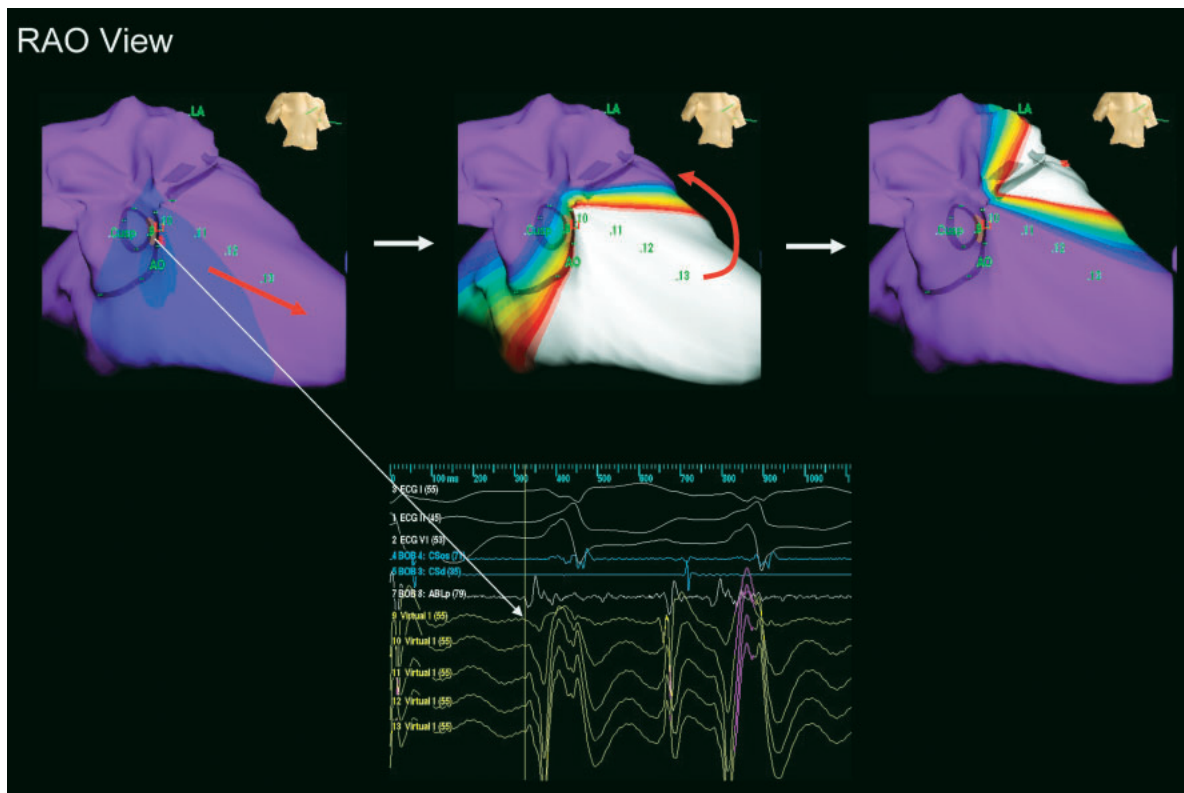


Figure 2 Dynamic wavefront map during the ventricular tachycardia (VT).

The VT focus was detected at the left coronary cusp (LCC). The virtual unipolar electrogram corresponding to the LCC exhibited a QS pattern, suggesting that the VT focus was located at the LCC.

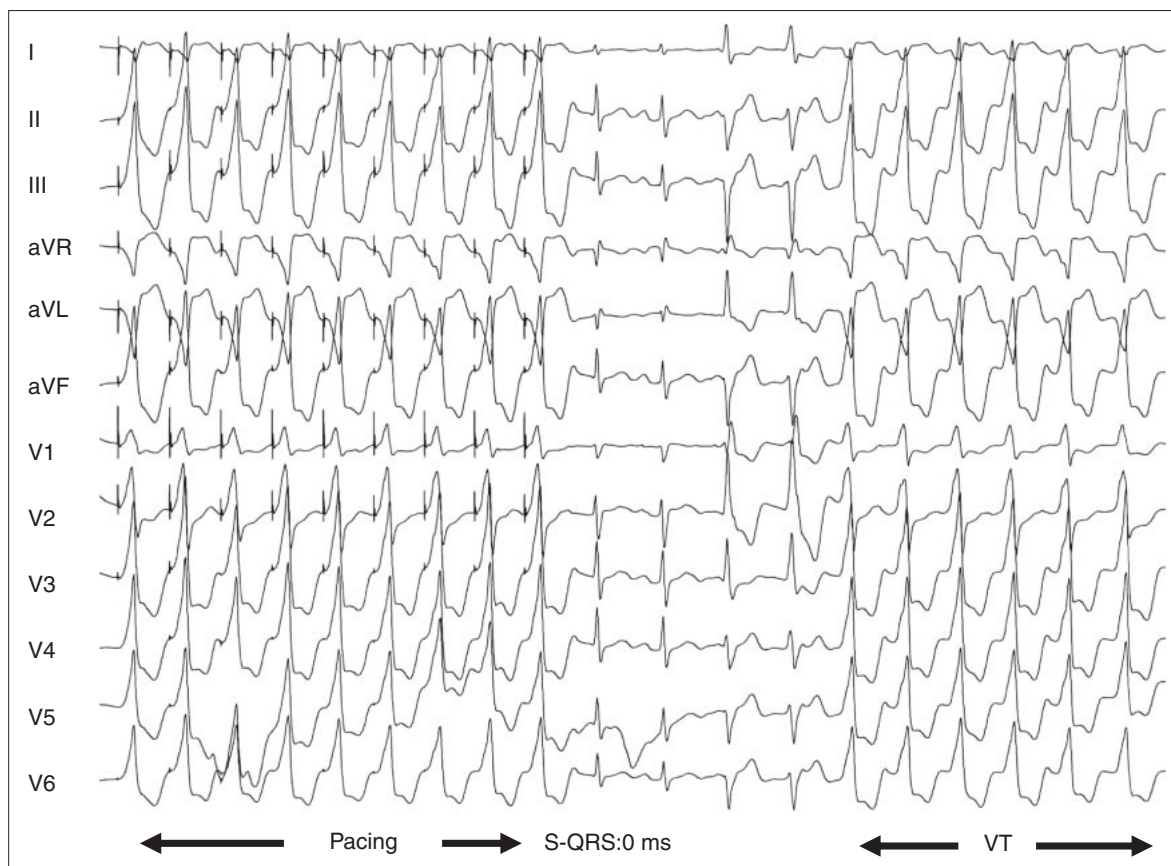


Figure 3 Pace mapping from the ablation site.

A perfect pace match was obtained at that site and the time from the stimulus to the QRS wave was 0 msec.

be due to a focal discharge or reentrant mechanism, depending on the case.⁹⁾ The present VT seems to have been due to a focal discharge mechanism rather than reentry because ventricular pacing did not initiate or terminate the VT, and ventricular pacing during the VT did not exhibit any entrainment phenomenon. The dynamic activation map created with the NCM clearly revealed the VT focus and subsequent activation to the entire LV, and the detailed analysis of the VUE enabled us to identify the precise localization of the VT focus. These features of the NCM facilitated a quick identification of the ablation target, leading to the easy elimination of the VT.

The validity and efficacy of the NCM has been reported not only for VTs¹⁻³⁾ but also for atrial tachyarrhythmias.⁴⁻⁸⁾ In our patient, the timing and morphology of the VUE at the ASC were the same as those of the contact unipolar electrogram, verifying the strong correlation between the virtual and contact unipolar electrograms.

The distance of the site from the center of the MEA to the target site has been reported to be a

crucial factor for the correct and precise analysis of the NCM recordings, and should be <4.0 cm in order to make a correct analysis.^{10,11)} In our case, the site of the VT focus was expected to be the ASC or superior mitral annulus based on the QRS morphology analysis. Before the RFCA, we had two options to introduce the MEA into the LV, which were the transaortic and transseptal approaches. If the MEA had been introduced into the LV by the trans-aortic approach, the distance from the center of the MEA to the VT focus would probably have been >4.0 cm. In contrast, if the transseptal approach was chosen, the distance could be <4.0 cm. However, if the MEA was located entirely within the LV, the MEA would be too far away from the VT focus. Therefore, after the introduction of the MEA into the LA via the transseptal approach, the MEA was slightly advanced from the LA to the LV so as to locate one forth of the MEA at the junction between the LA and LV, which allowed us to locate the MEA close to the VT focus.

Although safety and efficacy in manipulating MEA in the LV has been reported previously¹⁻³⁾

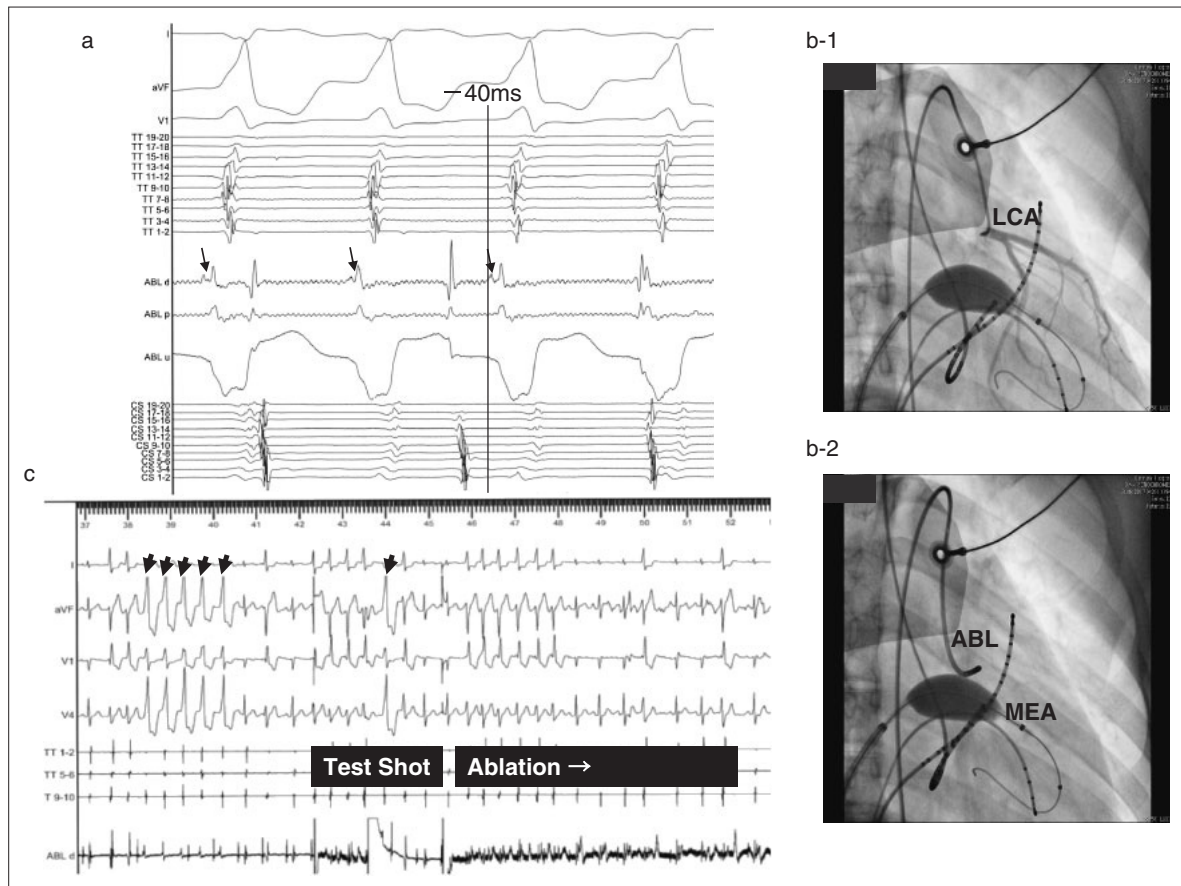


Figure 4

a: Local contact bipolar and unipolar electrograms at the ablation site. In the contact bipolar recording, a pre-potential (arrow) was detected which preceded the surface QRS wave by 40 msec. The contact unipolar recording exhibited a QS pattern, with an initial dull component and secondary sharp component.

b: Fluoroscopic view (RAO) of the ablation site. The ablation site was located at the left coronary cusp (LCC), which was distant from the ostium of the left coronary artery. Note that the noncontact catheter was located at the anterosuperior mitral annulus, which was positioned within 4 cm of the LCC.

c: The electrogram during the ablation. The clinical ventricular tachycardia (arrows) terminated during the test radiofrequency application and no acute recurrence was documented even under an isoproterenol injection. ABL: Ablation catheter, MEA: Multielectrode Array, LCA: Left Coronary Artery

and no procedure-related complications, which include cardiac tamponade, thromboembolism or valve/chordae injuries, have occurred in our 240 consecutive cases in which the MEA was introduced into the cardiac chambers, in order to avoid the procedure-related complications mentioned above, caution should be exercised during introduction and placement of the MEA into the LV, and ACT should be monitored constantly. In this patient we measured NIBP every 5 minutes to monitor hemodynamic status after deployment of the MEA, but in patients with unstable VT, direct arterial blood pressure monitoring would be strongly recommended.

In the left ASC, the electrogram-QRS interval was different from the stimulus-QRS interval, which

were -40 msec and 0 msec, respectively. If the left ASC was locally captured with a minimal output, both time intervals must be the same. The difference in these intervals might be due to the strength of pacing output of 9.9 V, which might have captured not only the true VT focus but also the endocardial exit of the VT, leading to unexpected shortening of the stimulus-QRS interval.

In conclusion, the dynamic activation map and detailed analysis of the VUE using the NCM was useful for identifying the VT focus and for examining the subsequent activation, and for navigating the ablation catheter to the target site. The RFCA of idiopathic VT originating from the ASC under navigation using the NCM was useful and safe in this case.

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